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DEFORMITY OF THE HAND AS A SYMPTOM.

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IT was when studying the bewildering array of chronic invalids under Prof. Charcot's care at the Salpêtrière that the following article was conceived and the material for its composition, in great part, collected. Not long after, the writer met with the excellent thesis of Meillet,[1] which has been of inestimable value as the only book of reference on the same subject.

An article on this subject may well be preceded by a few words on the normal position and appearance of the hand. The normal position of the hand depends on two important factors; first, the extreme mobility with the force of gravity, and, secondly, the resultant of the tonicity of antagonistic muscles. Each group of muscles moving the hand and fingers has its direct antagonist in some other group. Thus, flexors antagonize extensors; pronators antagonize supinators, and abductors antagonize adductors. Living, healthy muscle always has a certain tonus, which, together with the tonus of its antagonist, keeps the part moved by these muscles steady and ready at the shortest notice to obey the impulse of the will or of reflex excitation.

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[1] Des Déformations Permanentes de la Main au point de vue de la Séméiologie Médicale, par H. Meillet, *Thèse de Paris*, 1874.

The best example of this is the head, which, during waking hours, is balanced by the conjoint action of the neck muscles, but the moment the muscles lose their tonicity at the onset of sleep the equilibrium is destroyed and the head obeys the laws of gravity and nods. As in the neck, so in the hand the muscles keep it in a certain attitude.

The usual attitude of the human hand at rest is that of pronation and slight flexion of the wrist and semiflexion of the fingers, while the thumb is dependent and slightly inverted, so as to make its palmar aspect face very nearly the pulps of the fingers.

Now, this normal attitude of the hand may be changed in four ways.

1. A certain muscle or set of muscles may undergo atrophy, whereupon the antagonists of the same, which retain their tonus, will distort the hand in various ways.

2. Nearly the same thing occurs when a muscle or set of muscles is paralyzed without atrophy.

3. A spasmodic state of certain muscles may distort the hand by destroying the equilibrium of the muscular groups.

4. Anatomical, pathological changes may take place in the framework of the hand itself, through which vicious attitudes may arise.

Thus deformities of the hand may be, for convenience, divided into four classes.

1. Atrophic deformities.
2. Paralytic deformities.
3. Spasmodic deformities.
4. Deformities of purely local causation.

I.—ATROPHIC DEFORMITIES.

Under this head will be first described the hand of progressive muscular atrophy. The fully-developed muscular atrophic hand presents the deformed condition called commonly "main en griffe," or the claw-like hand. This deformity is by no means suddenly acquired, and in many cases of progressive muscular atrophy is never reached. In almost all cases, however, atrophy invades the hand somewhat, and a more or less perfect "main en griffe" results. When the disease invades the hand the patient notices that the hand easily tires and its capacity for finer movements is impaired. Soon the patient notices a falling away of the thenar eminence, and, from this time on, the atrophy extends, involving finally both thenar and hypothenar eminences, the interosseous and lumbrical muscles. The atrophy of the muscles of the thenar eminence and of the first interosseous space causes the thumb to separate from the hand and rotate on itself so as to make its palmar surface look in nearly the same direction as that of the fingers instead of in the opposite. This, together with the loss of the hypothenar eminence, gives a flat look to the hand, which causes it to be called "la main de singe," or the monkey hand. When the atrophy has reached and destroyed the interosseous muscles the typical "main en griffe," or claw hand, results. The trapezium and uniform processes of the carpus stand out in relief. The normal thenar and hypothenar eminences have given way to hollows in which the angular outlines of the first and fifth metacarpal bones are readily made out. The palm is hollowed out and very concave, and the interosseous spaces, instead of bulging as they should in the well-formed hand, are sunken and show the outlines of the metacarpal bones on both the dorsum and in the palm.

The first metacarpal bone is drawn toward the same level as the others, and is rotated outward on its longitudinal axis. Moreover, the antagonists of the thenar muscles cause the thumb to assume still further a vicious attitude. The extensor ossis metacarpi pollicis draws the metacarpal bone out, while the flexor longus pollicis being stronger than the extensor secundi internodii pollicis, the two phalanges of the thumb, especially the second, tend toward flexion, thus giving the thumb very much the appearance of the fourth toe of birds.

The atrophy of the interosseous and lumbrical muscles leaves no antagonists to the long extensors and flexors, hence the former extend the first phalanges, while the latter flex the second and third.

Together with the deformity of the hand its intrinsic movements are restricted. Abduction, adduction, and opposition of the thumb are impossible. Flexion of the first and extension of the second and third phalanges, and ab- and adduction of the fingers are impaired in proportion as the atrophy of the interossei and lumbricales is partial or complete.

When these muscular changes have existed a long time there ensues a change in the fibrous elements of the hand, which seems to fasten it in this unnatural position and to prevent, even by the exercise of considerable force and the production of a great deal of pain, even a temporary return to its normal shape.

The description of the "main en griffe" as above given is by no means a constant sequel of progressive muscular atrophy, and for the following reasons. It is rare for the atrophy to be complete in the hands before it invades other parts, and if the long extensors and flexors of the fingers in the forearm are atrophied a typical claw hand cannot result.

Again, the atrophy may commence in some other part of the body, and the patient may die with the hand nearly normal or only slightly involved. Hence we more often see the "main de singe" (monkey hand) in progressive muscular atrophy, while the typical "main en griffe" is better exemplified in some cases of nerve injury or disease, as in leprosy.

The points in the differential diagnosis between this deformity and others resembling it are, first, the retention, to the last almost, of farado-contractility of the muscles; secondly, the common involvement of both hands; thirdly, the existence of atrophy in some other part of the body; fourthly, the progressive tendency; fifthly, the absence of anæsthesia, and also the clinical history. From the atrophy following any other spinal lesion or any cerebral lesion it is distinguished by the fact that it is preceded and accompanied by no paralysis or anæsthesia. From the atrophy following nerve injury it is distinguished by the facts, first, that its atrophy has no regular distribution, while that following nerve injury is confined exclusively to the physiological distribution of the injured nerve; secondly, because it was preceded by no paralysis and accompanied by no anæsthesia. From the atrophy following neuritis it is told by reason of the absence of pain and anæsthesia, and by its irregular distribution, the atrophy in neuritis, as in nerve injury, being localized. From the occasional atrophy following lead-poisoning it is distinguished by the absence of paralysis, by the electrical reactions, and by the absence of a history of plumbism. From the atrophic hand of pachymeningitis cervicalis and leprosy it is easily told by its clinical history.

The history of the patient, whose hand is represented, is here inserted, as it is, in the most important details, a typical history.

The patient was Marie A. T——, St. Alexandre Ward of the Salpêtrière Infirmary[1]; 40 years old; a maker of fringes. Her occupation called for continual use of the hands and the pressure of a round handle in the palm of the hand a good deal. About a year ago she noticed that when her hands were a little chilled it was very hard work to use them unless she rubbed them very hard.

Very soon she noticed decided loss of power in the hands, and asserts that at this time there was slight formication in the parts. Next she noticed a wasting of the thenar eminence of both hands, more marked in the right. A little later the hypothenar eminences began to fall away, and the interosseous spaces became sunken, and the hand gradually assumed a claw-shaped appearance. Of late shoulder movements have become difficult. She denies ever having had pain.

May 17, 1880. Present condition: Atrophy of muscles of hand, forearm, arm, and shoulders. More marked, however, in the hands, whose thenar and hypothenar regions are very flat, and whose interosseous spaces are sunken. The left hand, in particular, has assumed a partial "main en griffe" attitude. (See fig. 1.)

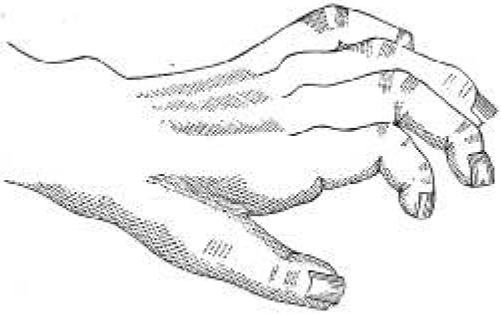

FIG. 1.—Left hand of Marie A. T——. A case of common progressive muscular atrophy.

The thumb is abducted and drawn back on a plane with the rest of the hand, while the last phalanx is flexed. The first phalanges of the fingers are slightly extended, while the second and third are slightly flexed.

Voluntary movements of the atrophied parts are very restricted, especially those of abduction and adduction, extension of the last phalanges of the fingers, and opposition of thumb. All vigorous efforts in that direction result in increased extension of the first and flexion of the two last phalanges. Complete closure of the hand is also impossible. As yet no rigid fixation has taken place, so passive movements can be made, although they are rather painful to the patient.

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[1] Service of Prof. Charcot.

There is no impairment of sensibility, no tendon reflex in the hands, and an abnormally marked one at the knees.

Lower extremities are intact, and all the bodily functions are normal.

The next atrophic deformity of the hand described will be that of leprosy—morphœa alba or lepra anæsthetica. The hand of this disease when it is fully developed is a typical "main en griffe." The hand is entirely deprived of its fleshy covering, and the bony prominences and tendons everywhere show through the tightly drawn skin. The thenar and hypothenar eminences have vanished, the interosseous spaces are sunken, and the palm is extremely hollow. The hand is slightly extended, the thumb is rotated outward, its metacarpal bone is extended and its two phalanges flexed. The first phalanges of the fingers are extended, while the second and third are generally flexed, and in an advanced stage completely and tightly flexed. The long flexor tendons in the palm are very prominent and extremely tense. The hand is held very rigidly in this attitude, and, as might be well imagined, its intrinsic movements are almost, if not entirely, abolished. It is in this deformity of the hand, in particular, that some change takes place in the fibrous and articular apparatus of the hand which renders a restoration to its original attitude, even by force, almost impossible. With these atrophic changes there are apt to be ulcerative or tubercular affections about the fingers, and an impairment or, in many cases a complete loss of general sensibility in the parts. This form of leprosy, which receives its name because when fully developed the patient presents on various parts of his body patches of skin, large or small, generally white, which are entirely devoid of sensibility, is endemic chiefly in Egypt and Arabia, but is also known in almost all equatorial countries; also in more temperate countries, as Turkey, Greece, China, and even in some high latitudes, as in Norway and Sweden. In our country and France only imported cases are seen, and it was from an Egyptian who attended Prof. Charcot's clinic that the following history and sketches were taken. For the history I depend on my own notes taken at the time and a full account of the case which appeared in the *Progrès Médical*, Dec. 25, 1880, reported by Ballet, interne at the Salpêtrière.

The patient, H. F., male, twenty-four years old, was born at Cairo, Egypt. He had no hereditary taint. The disease commenced at the age of eighteen. For many years before the patient had led a very dissipated life. He drank regularly about two litres of *raki* a day, besides a large quantity of wine, cognac, and absinthe; he consumed, besides, an enormous quantity of hashish; he smoked about fifty cigarettes a day, each containing fifty centigrammes of Indian hemp, and ate a pastile of the extract, weighing 1.50; he also carried sexual intercourse to a wonderful excess. The disease commenced by anæsthesia of the lower extremities; then the head and neck lost sensibility, and then the neighborhood of the nipples. Two years later the hands commenced to atrophy. Examination of the patient reveals the existence of large placques surrounded by a slightly elevated border, which is strongly pigmented. The skin in these zones is completely anæsthetic. One comprises the head and neck, two more each arm and shoulder, a small one surrounds each nipple, while another comprises the external genital organs. A very pronounced atrophy affects all the muscles underlying the anæsthetic areas.

The atrophy is very marked in the feet, legs, forearms, and hands, and also in the face, particularly the orbicularis palpebrarum, occipito-frontalis, and zygomaticus major.

This facial atrophy gave a peculiar facies, as there was inability to close the eyes tight, and the atrophy of the oral muscles gave the patient a particularly woe-begone expression. There was an ulcerative keratitis with pannus in both eyes. As for the hands, which concern us particularly, they had lost about all their intrinsic muscular substance (see fig. 2), the thenar and hypothenar eminences had disappeared, and the interosseous spaces were very hollow. The hand had assumed an almost typical "main en griffe" attitude, and by local changes was pretty tightly held in this position. The intrinsic movements of the hand were of course impossible. The patient could write his name by holding the pencil stiffly between his index and middle fingers, and by moving the hand as a whole.

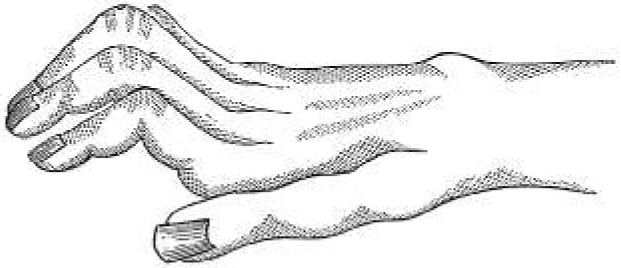

FIG. 2.—Right hand of H. F. Case of morphœa alba.

The results of a careful electrical examination by Dr. Vigouroux showed that there was no response to either current where the atrophy was well marked, but that in a few muscles, as the peronei and the pyramidalis nasi, there was degeneration reaction to the galvanic current.

This deformity of the hand is easily distinguished from any other by the history, by the anæsthetic patches on the body, and particularly by the anæsthesia of the hands.

In myelitis of the anterior horns in the cervical region of the cord we have often a condition of the hands which, perhaps more nearly than any other, resembles the hands of muscular atrophy. Like it the atrophy is generally bilateral, and generally accompanied by no impairment of sensibility of the part; but unlike muscular atrophy that of myelitis is preceded by paralysis, and generally attacks groups of muscles or whole extremities, while muscular atrophy may invade and destroy a part of a muscle and leave the rest intact. In the hand of cervical paraplegia, also, we get a loss of farado-contractility and a degeneration reaction to galvanism.

The deformity resulting from myelitis of the anterior horns varies from a simple atrophy of the larger groups of muscles to a total atrophy of all the hand muscles. When the thenar eminence is atrophied we get simply the monkey hand, which is quite common, and when the other intrinsic muscles are involved there results a more or less complete "main en griffe," modified in many cases by a paralysis or paresis of the long flexors or extensors in the forearm.

The deformity of the hand, resulting from a common myelitis, differs in no way from that of myelitis of the anterior horns, except it is accompanied by anæsthesia. The man whose history and a sketch of whose hand is annexed, had a cervical myelitis chiefly confined to the anterior horns, but undoubtedly implicating the sensory zone somewhat, as evidenced by the sensory disturbance in one of his arms. His history was as follows:

P. C., male, forty-eight years old,[1] March 30, 1880, when intoxicated, went to sleep on the floor of a cold room, in a draught. He was perfectly well at 12 o'clock, when he fell asleep. At 4 A. M., when he awoke, he found his arms were powerless from shoulders down, and that he had no sensibility from just above the elbows down to the fingers. Two weeks later he could move the right index finger slightly, and at the same time sensibility had gradually reappeared in the whole arm, last in the right medius, which was also the last to regain its power of motion. When first seen, June 2, 1880, the patient could use his right hand a little, but there still remained a slight amount of numbness and sense of powerlessness. The left arm was quite helpless from the shoulder down, and there was no sensibility from a little above the elbow to the finger tips. The muscles were flabby and degenerated, and when asked to raise the arms only the trapezius acted on either side. There was the reaction of degeneration in some of the muscles of the right upper extremity and in almost all of the left upper extremity. He was treated systematically with electricity for months and nothing new developed, except the knowledge that he had been subject to fits of psychical epilepsy for years.

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[1] Service of Dr. Seguin, Manhattan Hospital.

At the time the accompanying sketch was made (see fig. 3) the right arm, forearm, and hand had entirely recovered, and the condition of the left was as follows: There still remained some atrophy in the suprascapular muscles. The arm muscles were nearly normal, but the forearm muscles responded poorly to faradism, and with a slight degeneration reaction to galvanism. Voluntary motion was possible in all parts above the wrist. There was no voluntary contraction of the intrinsic muscles of the hand, and in fact little of these muscles remained. Thenar and hypothenar eminences were gone, and the interosseous spaces were sunken. The extensor tendons stood out on the back of the hand, while in the palm the long flexor tendons were very salient. The position assumed was rather that of the "main de singe" than the "main en griffe." The thumb had receded to the plane of the fingers, but the first phalanges, except those of the ring and little fingers, were not so much extended as is usual in the claw hand. The joints were still mobile. Whatever reaction is present at all in the hand is a well-marked degeneration reaction to a

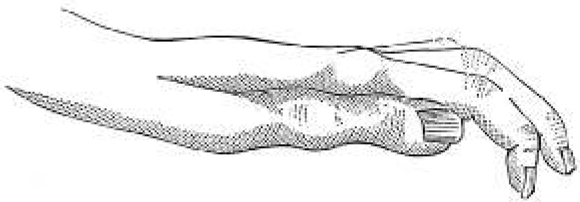

FIG. 3.—Left hand of P. C. Case of cervical paraplegia.

strong galvanic current, although at times a slight response has been noticed to a very strong faradic current in the first interosseous muscle.

Sensibility is still poor in the forearm and almost nil in the hand, only the strongest faradic current being felt. Very often the galvanic application he now has (negative pole, labile) will bring out on the left arm and forearm a beautiful urticaria rash which lasts only a few minutes. At other times a fine, red, papular eruption follows the same application.

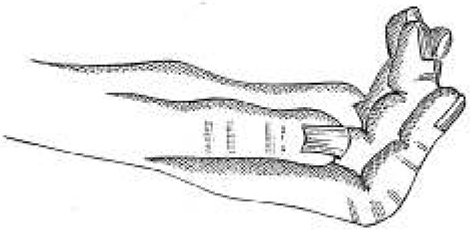

FIG. 4.—Right hand of case of cervical paraplegia in the service of Prof. Charcot at the Salpêtrière.

In amyotrophic lateral sclerosis there is generally first a spasmodic and then an atrophic deformity of the hand; but as the atrophic deformity is most often seen and more permanent it will be introduced here. The course of the disease is so typical that only a *résumé* of its symptomatology need be given. The patient complains of weakness, formication, numbness, or perhaps muscular pains of the upper extremities. Fibrillary twitchings, atrophy, and progressive paralysis soon follow. There may be, and generally is, before the paralysis and atrophy have advanced far, a contracture of the hand and fingers due partially to unopposed action of muscle still remaining healthy, but also to a true spasm of some muscular groups. There is adduction of the arm, semiflexion and pronation of the forearm, semiflexion of the wrist and fingers. Later in the course of the disease, after the atrophy and paralysis have progressed, this spastic condition generally relaxes, sometimes completely, and there remains the " main en griffe " or " main de singe." At a variable length of time after the onset of symptoms in the upper extremities walking becomes difficult. The legs feel heavy, the feet drag, and the patient frequently falls. There exists, particularly in the legs, a muscular hyperæsthesia, and the patient has attacks of "spinal epilepsy," brought on by movements or contact, and consisting of jerkings, adduction of the thighs, and crossing of the feet and legs. Soon now the patient becomes bed-ridden, and the stiffness of the legs becomes permanent. Paralysis and muscular atrophy soon set in but are never so marked as in the arms. The head is bent forward, often so that the chin touches the sternum, and the spine is very rigid. At last labio-glosso-laryngeal paralysis sets in and death soon results.

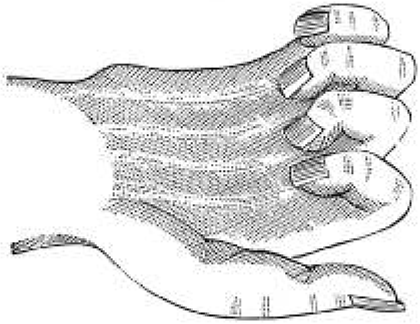

FIG. 5.—Right hand of Louisa ——. Case of amyotrophic lateral sclerosis.

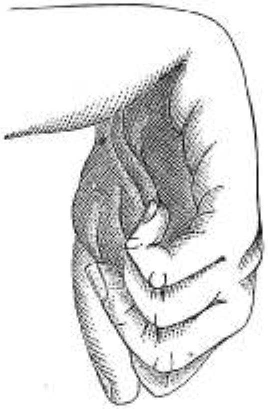

FIG. 5½—Forearm and hand of Charcot's case of amyotrophic lateral sclerosis.

The patient from whom fig. 5 was taken had entered the third and last stage of the disease, and her hand, as figured, was more atrophic than spastic. She was an embroiderer, æt. 49, and entered the St. Luke ward at the Infirmary of the Salpêtrière Nov. 13, 1878.[1] This case had followed the typical course, and her condition when the hand was figured was as follows:

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[1] Service of Prof. Charcot.

She was bedridden. The face had a grinning demented look. Eyes staring, brows elevated, corrugators contracted, mouth half open, lips drawn and stiff; saliva dropped almost constantly from the mouth. Tongue was incapable of protrusion, thick and rough; articulation was impossible, deglutition was difficult, regurgitation through nose, and frequent choking fits occurred. The head was pretty rigidly flexed on the chest. The upper extremities were not very rigid, but helpless; the lower extremities were rigidly extended, adducted, with the feet in extreme talipes equinus. The sensibility was good, voluntary motion was almost nil, and the reflexes enormously exaggerated all over the body. The patient was very emotional, and simpers and cries easily. The atrophy was chiefly confined to the upper extremities. The hands, especially the right (fig. 5), were very much atrophied. All the fleshy eminences were gone, the palm was very hollow, and the fingers were spasmodically flexed into the palm. The hand itself, unlike Charcot's case, was about on a line with the forearm.

The only hand that amyotrophic lateral sclerosis could be confounded with, is that of progressive muscular atrophy or cervical paraplegia. The rapid progress of the disease and the existence of a spasmodic element would exclude the former, while the absence of complete paralysis and the preservation of farado-contractility of the muscles would exclude the latter disease.

The clinical history of the disease is so typical that a mistake is scarcely possible.

To exemplify the deformity of the hand which results when the spasmodic element predominates, there is inserted a figure 5½ from Charcot,[1] which he describes (p. 235) thus: "The arm is adducted and the shoulder muscles resist abduction. The forearm is semiflexed and pronated; supination and extension are difficult and painful. The hand is semiflexed and the fingers are flexed on the palm."

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[1] Leçons sur les Localisations dans les Maladies du Cerveau, etc., Paris, 1876, p. 234.

In the hypertrophic cervical pachymeningitis of Charcot a peculiar deformity of the hand results, a description of which, as the writer has never seen a case, will be presented as given in the writings of Charcot, Jeoffroy, and Hallopeau.

The disease is generally divisible into two stages—the painful period and the paralytic and atrophic period. The disease commences by severe pains in the occiput and back of the neck, much aggravated by pressure on the spinous processes. These pains often radiate over the head, down the back, and down the arms, and there are, besides, frequent rheumatoid pains in the shoulders and elbows. The neck is kept rigid as in Pott's disease.

With these pains the patient complains of formication, numbness, and perhaps of some anæsthesia in patches, in the hands particularly. Bullous or pemphigus eruptions are sometimes seen. There are cases, however, in which the pain is chiefly peripheral, and confined almost entirely to the articulations. Sometimes a true remission intervenes between the two periods. This remission is not constant, however, and the two periods merge one into the other.

After a longer or shorter painful stage, two to three months, the patient notices a weakness and uselessness of one or both upper extremities. Very soon an atrophy commences in the hands and extends to the forearms. The muscles often more paralyzed and atrophied are the intrinsic hand muscles and the flexors of the fingers and hand. The pronators of the forearm generally suffer with the flexors. This leaves the extensors and supinators the only healthy muscles, and their unopposed action produces the deformity of the hand named by Charcot "la main du prèdicateur emphatique," which presents the following characteristics: The hand is extended and supinated. At times all the phalanges are somewhat flexed. At other times the fingers are extended. The thumb is applied to the side of the hand and slightly flexed. The hand itself is almost devoid of muscular covering. This atrophy and paresis generally soon invade the thoracic muscles, and in this way sometimes causes death. Of course when the secondary descending changes invade the lower cord, the lower extremities may become paraplegic with vesical and rectal complications, or tetanoid symptoms will develop as in descending degeneration from other causes.

This disease can be hardly confounded with any other, and may with certainty be distinguished from amyotrophic lateral sclerosis by the history of the painful stage, by the disseminated patches of anæsthesia and the occasional eruptions, and also by the entire absence of bulbar symptoms. The accompanying figures are copied from the works of Charcot, Jeoffroy, and Meillet.

The following is a *résumé* of the case whose hand is depicted, taken from the monograph of Jeoffroy. (See fig. 6.)

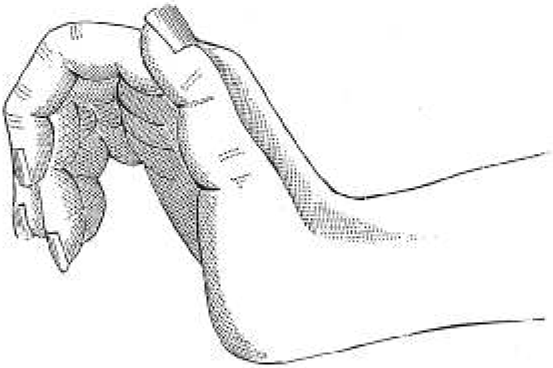

FIG. 6.—Right hand of Charcot and Jeoffroy's case of hypertrophic cervical pachymeningitis.

On Aug. 6, 1865, this woman, 29 years old, was exposed to cold and wet. During the next 48 hours she had repeated chills. At that time sharp pains commenced in both right extremities, chiefly in the course of nerves and in the joints.

In the middle of September the pains invaded the left side. At this time fibrillary movements were noticed in the right upper extremity.

Two weeks later there was noticed a difficulty in raising the right arm, in opposing the thumb and in ad- and abduction of the fingers. Atrophy had already set in in the region of the deltoid and in the intrinsic muscles of the hand.

In April, 1866, the right elbow was semiflexed, the forearm semipronated, the wrist semiflexed, and the fingers flexed in on the palm. This contracture could be voluntarily overcome by an effort of the will.

A month later contracture appeared on the left side. There were diminished electro-contractility and lowered temperature in the affected parts. The 12th of December all contracture had disappeared, and paralysis of the previously contracted muscles was marked.

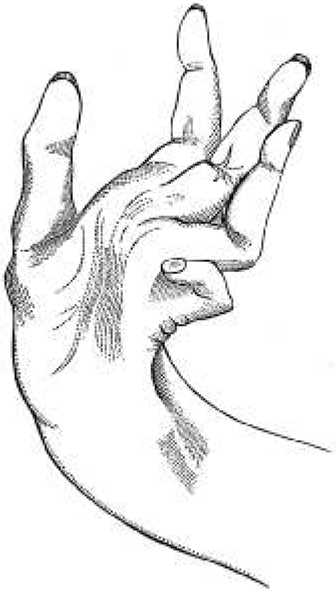

FIG. 7.—Left hand of another case of hypertrophic cervical pachymeningitis under Prof. Charcot's care. Taken from Meillet's monograph.

Early in 1868 the patient came under the care of Prof. Charcot at the Salpêtrière. She was very emaciated and bed-ridden from weakness and contracture of the legs. The left upper extremity was atrophied and flaccid, the right hand was extended at a right angle to the forearm, and the thumb was also extended, except the terminal phalanx, which was semiflexed. The fingers which could still be extended are flexed on the palm. The forearm was flexed on the arm and the arm was adducted. All voluntary movements were abolished. There were some fibrillary contractions in the left hand. There was much anæsthesia, more marked on the right side. Electro-contractility was preserved, but quantitatively changed.

As deformities of the hand resulting from disease or injury of a peripheral nerve present many of the same characteristics a few words will be said about them together, and then special mention will be made about each variety.

Whether the nerve be injured by a neuritis, a neoplasm, by prolonged pressure, by bruising, laceration, or section, and the injury result in a local disorganization, or any change which completely destroys the conductibility of the nerve fibres, the same results ensue. First, of course, there is a paralysis[1] and anæsthesia in the peripheral distribution of the injured nerve bundle. Next the muscles supplied by the nerve, being cut off both from their motor and trophic centres, atrophy, if the separation be complete, entirely; and, lastly, changes take place in the skin of the affected part, due to the anæsthesia and the cutting off of the trophic supply.

The limitation of all these changes to the exact distribution of the nerve injured, distinguishes the deformities due to changes in a peripheral nerve from any other. If regeneration of the nerve take place soon, the part may return to its normal condition, but when regeneration is slow or absent, degeneration of the muscular substance ensues, and we find they respond in an abnormal manner to galvanism. The trophic changes occurring after nerve injury are various. The skin is thin and smooth. The fingers are often clubbed, and the nails curved and frequently rough and brittle.

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[1] The temporary deformities caused by the paralysis of the first stage of nerve section, etc., will be mentioned when speaking of paralytic deformities of the hand.

If there be much anæsthesia, as there usually is, the patient will frequently mechanically hurt the anæsthetic parts in various ways, or burn or freeze them, and troublesome indolent ulcers will result.

The distribution of the affected nerve is often the seat of sharp, tearing or burning pains, which are excessively harassing to the patient. One of the most common atrophic deformities of the hand due to nerve injury is that caused by contusion at the elbow, or by section of the ulnar nerve at the wrist. The ulnar supplies, in the forearm, the flexor carpi ulnaris and the two internal portions of the flexor profundus digitorum; in the hand, all the muscles of the hypothenar eminence, the adductor pollicis, the inner head of the flexor brevis pollicis, the two inner lumbricales, the two inner interosseous spaces entirely, and the others either alone or in conjunction with the median nerve. Variations from this distribution are uncommon but not unknown.

The deformity resulting from a destructive lesion of the ulnar nerve at the elbow, or above is called the ulnar griffe (griffe cubitale of the French). The griffe consists, first, in marked prominence of the unciform, pisiform, and third and fourth metacarpal bones, owing to an atrophy of the muscles of the hypothenar eminence, the interosseii, and two inner lumbricales. Secondly, in some want of fulness about the first interosseous space and thenar eminence, owing to the atrophy of the adductor pollicis and the inner head of the flexor brevis pollicis. The vicious attitude the hand assumes is first, perhaps, a slight extension of the hand on the forearm, with a slight depression over the site of the flexor carpi ulnaris and that of the outer half of the flexor profundus digitorum in the forearm. The first phalanges of the ring and little fingers are slightly extended, because the long extensors of the fingers are no longer resisted by the lumbrical muscles.

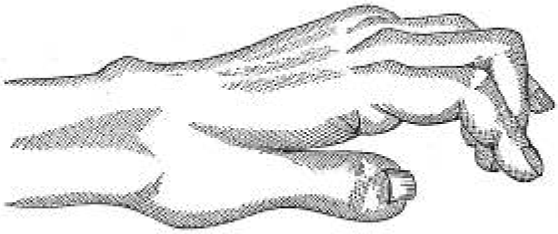

FIG 8.—Left hand of patient E. H., with section of the ulnar nerve at wrist.

Their third and second phalanges are flexed by the flexor sublimis digitorum, there being no antagonists where the third and fourth interossei are paralyzed and atrophied.

The medius has generally much the same deformity as the annulus, but the index often has its normal position and motion, because its intrinsic muscles are, in most hands, supplied by the median nerve. The thumb retains its power of opposition while it has lost that of adduction and partially that of flexion.

After injury to the ulnar at the wrist the same state of things exists except there is no paralysis of the flexor carpi ulnaris, or of the outer half of the flexor profundus digitorum. The presence or absence of this paralysis has little effect on the resulting deformity, and so we see in all cases of ulnar injury a sort of "main en griffe," with contracture more marked in the ring and little fingers.

The following case came to the clinic of Dr. Seguin at the Manhattan Hospital, March 2, 1881. (See fig. 8.)

E. H., a janitress, forty-nine years old. Nine months before, while washing windows, she pushed her left hand through a thick pane of glass, and on the sharp, fractured edge cut the flexor ulnar side of her forearm, near the wrist, to the bone. Immediately after the injury she noticed a prickling sensation in the ring and little fingers. Later they became swollen and painful. The cut healed in two weeks. At the expiration of that time, when the hand was taken off the splint, the patient asserts that the hand was flat and the fingers crooked. On examination it was seen that the eminences were flat. The two distal phalanges were flexed. The thumb was abducted and semiflexed. The hand was somewhat swollen and the skin in great part smooth. The finger tips, particularly that of the annulus, were clubbed and the nails curved. There was almost complete paralysis of the intrinsic muscles of the hand. There was fair sensibility to painful impressions in the distribution of the ulnar nerve, but tactile sensibility was very poor. There was no reaction in any of the hand muscles to faradism, and degeneration reaction in all except, perhaps, in those of the second interosseous space, where An C C and Ca C C were about equal.

Injury to the median nerve at the wrist results in a deformity of the hand, of which the following case is so perfect an example that it will be immediately introduced. (See fig. 9.)

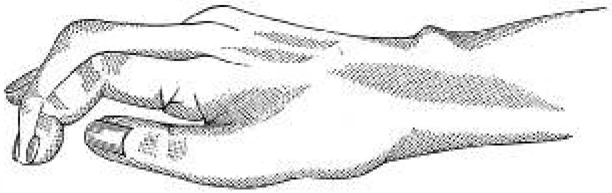

FIG. 9.—Right hand of C. K., with section of median nerve at the wrist.

C. K., a laundress, twenty-two years old, came to the clinic of Dr. Seguin at Manhattan Hospital, May 11, 1881. Three months before, while washing, she pierced her right palm with a needle. Suppuration followed, and on the fifth day two incisions were made, the one on the anterior surface of the wrist without question severing the median nerve. Three weeks later she first noticed anæsthesia in the index and middle fingers and on the pulp of the thumb.

At the time of examination there were anæsthesia, smoothness of the skin, and marks of burns and injuries in the median distribution. There was some falling away of the thenar eminence, a tendency toward extension of the first phalanges of the index and medius, and flexion of their last two phalanges. The intrinsic movements of these fingers are limited, if at all present. The thumb is adducted and has lost its power of opposition. The ring and little fingers are in normal position and of normal appearance.

The deformity resulting from musculo-spiral nerve injury will be mentioned under the head of paralytic deformities.

In injury to the brachial or axillary plexus (see figs. 10 and 11) hand deformities result, varying of course according as all or only few of the cords of the plexus are injured. The manifestations are not apt to be restricted to the distribution of any one nerve, and the more common result of such injury is a general wasting and a common "main en griffe," as in the following case:

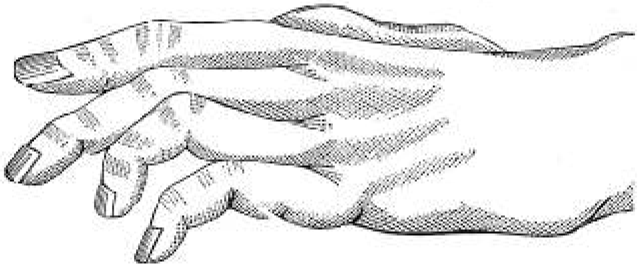

FIG. 10.—Left hand of Dr. Seguin's case of injury to the brachial plexus. Ulnar filaments chiefly involved.

A woman, forty years old, in the St. Jacques ward of the Infirmary of the Salpêtrière,[1] a tailoress by occupation, when twelve years old dislocated her right shoulder. Since then it has been dislocated six times. The last dislocation occurred two and a half years ago, and was treated at Lariboisière. Then the patient noticed for the first time that her hand was getting thin and that there was a very pronounced atrophy of that side. When examined, May 15, 1880, there was some rigidity of the right shoulder. No atrophy of the shoulder, arm, or forearm muscles. Apparently complete atrophy of all the intrinsic hand muscles. The thenar and hypothenar eminences were wanting, the interosseous spaces sunken, and the palm hollow.

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[1] Service of Prof. Charcot.

The thumb was extended and rotated inward, the first phalanges were extended, the second were flexed, and the third about straight.

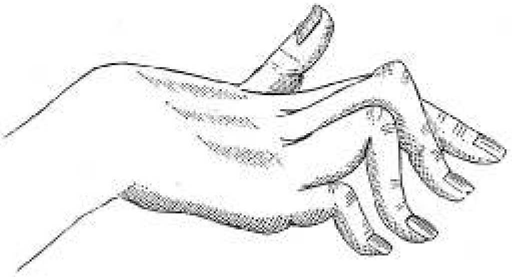

FIG. 11.—Right hand of Prof. Charcot's case of injury to the brachial plexus.

This case is not a typical one of nerve injury, as its course was not marked by pain or much impairment of sensibility.

The deformity resulting from neuritis differs in no way from that of nerve injury, but the clinical history is different, the severe pain particularly characterizing the first stage of neuritis.

The electrical reactions are the same as in nerve injury, and there commonly is anæsthesia.

K. G., a domestic, thirty-two years old, came to the clinic of Dr. Seguin at the Manhattan Hospital, Aug. 4, 1880.

For two years she had had occasional pain over brachial plexus (right side). She had had no injury to shoulder. May 11, 1880, she cut her right thumb. It bled little, but that night the thumb was the seat of much pain, which by the next night had extended to the palm of the same hand. Later the pain ran from the thumb up the flexor surface of the forearm, and then the fingers began to flex until they acquired the position now occupied by them, they never having relaxed. The severe pain lasted about three weeks, but ever since there have been occasional twinges in the hand. There was at one time considerable swelling of the palm, but there were no indications that any suppuration took place.

On examination it was seen that the fingers of the right hand were semiflexed, fixed, small, smooth, and provided with new nails. There was some atrophy of the eminences, and movements of fingers were restricted. (See fig. 12.)

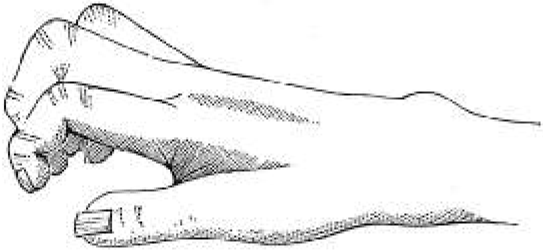

FIG. 12.—Right hand of K. G., a case of neuritis.

2.—SPASMODIC DEFORMITIES.

We now come to speak of spasmodic deformities of the hand, and of these the most common and well known is the permanent contracture of hemiplegics.

In a large proportion of cases of hemiplegia of cerebral origin, at the expiration of from one to three months, a variable degree of descending degeneration in the lateral columns of the cord has taken place. This lateral sclerosis is a lesion which irritates the cells in the anterior horns of the spinal corn, and in this way greatly heightens the reflex irritability of the spinal cord and increases muscular tonus on the paralyzed side. As a result of this certain muscular

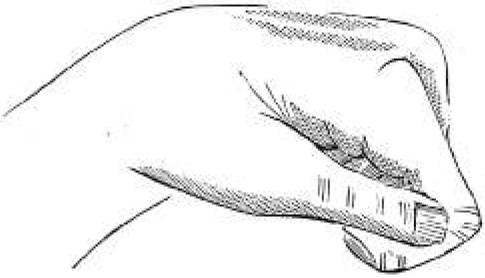

FIG. 13.—Right hand of a patient with common hemiplegia in the service of Prof. Charcot.

groups on the heretofore relaxed and paralyzed side, generally the flexors, pronators, and adductors in the upper extremity, and the extensors and adductors in the lower extremity, take on a spasmodic action which results in the condition called permanent hemiplegic contracture.

That it is a purely reflex condition is proven in many ways, but principally by the fact that the contracture is increased by the application of any local irritation to the part, cold, electricity, a rapidly vibrating body, pressure, pain, etc., etc., and by any effort to use the part, and that when the part is quiet and warm, as during sleep, the contracture may disappear, or at all events become less.

This contracture is, as a general thing, accompanied by no atrophy, and by only slight trophic changes in the part. As a rule it is permanent, but it sometimes spontaneously disappears and leaves the part flaccid.

Hemiplegic contracture of the hand varies considerably, but it generally consists in flexion of the hand, inversion and flexion of the thumb, and flexion of the fingers into the palm (see figs. 13, 14, 15). This contracture may vary in severity from a gentle shutting of the hand to the closure of the fist like a vice, and a deep indentation or puncture of the palm by the nails of the flexed fingers (see fig. 16). This spasmodic condition of the flexors can be overcome by the exercise of considerable force which gives the patient a

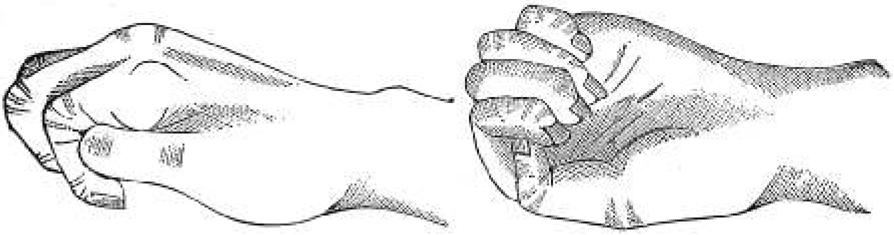

FIG. 14.—Right hand, and Fig. 15, left hand of common hemiplegics under Professor Charcot's care.

good deal of pain. Many hemiplegic contractures, as before said, relax of themselves when external irritations are reduced to a minimum, as during sleep. There is no atrophy or even emaciation. The hand is, as a general thing, well nourished, but, more from disuse than any thing else, the skin is generally smooth and thin; sensibility is, as a general thing, preserved, and reaction to faradic electricity is retained; there may or may not be complete paralysis in the contractured part.

As said above, a voluntary effort to use the part increases the contracture. Not only this, vigorous voluntary movements of the opposite hand, particularly flexion, also increase the contracture.

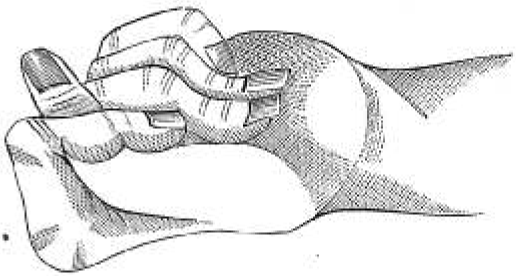

FIG. 16.—Left hand of a demented hemiplegic under Professor Charcot's care.

This constitutes the phenomenon known under the name of "associated movements," and is present in many cases of hemiplegia where no contracture exists. Hemiplegic contractures are so common that no time will be given to the narration of a case, but several figures will be introduced to demonstrate their different phases.

When a destructive cerebral lesion causing hemiplegia occurs in very early childhood, the limbs on the paralyzed side seldom, if ever, attain the same development as those on the other side. There may also be left a spasmodic or a paralytic deformity. Of the latter we will speak later on, of the former now. This peculiar deformity is designated as that produced by cerebral atrophy.

In the infant, as in the adult, hemiplegia generally develops contracture. The position is also generally that of pronation and flexion of the hand and flexion of the fingers. In young children the osseous, cartilaginous, and ligamentous structures of the wrist joint are soft and malleable, so to speak, and the result of a long continuance in a vicious position is a permanent change in the articular structures, rendering a normal attitude and movement of the hand almost impossible. The deformity of the hand resulting is generally as follows (see fig. 17). The forearm is semiflexed and partly pronated, the hand is strongly flexed on the forearm and is generally inclined more to the ulnar side, while the fingers are generally slightly, if at all, flexed on the palm.

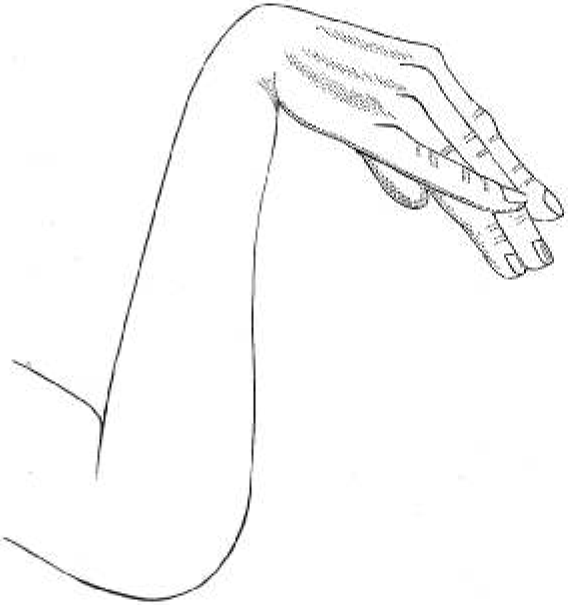

FIG. 17.—Right hand of John M——, Dr. Seguin's case of infantile hemiplegia and cerebral atrophy.

Extension of the wrist is almost impossible, the palmar edge of the carpal bones having become thin, and very likely the articular end of the radius being also bevelled off on its palmar aspect. The hand is smaller than its fellow, and may or may not show some general atrophy. Voluntary movements will be very restricted, while there may be athetoid movements and very surely associated movements in the deformed hand. Sensibility is generally preserved and electro-contractility (unless there be atrophy) normal.

The following case is typical:

John M., 21 years old, came to the clinic of Dr. Seguin, at the Manhattan Hospital, Aug. 9, 1881.

The mother states that soon after the birth of the child she noticed he had strabismus. When the child was about four months old she began to notice a weakness of the right arm and leg.

When three years old the child had two fits in rapid succession. Three years later he had another fit, and since then he has had typical epileptic attacks at intervals of six or eight weeks to the present time. The hand has been contractured for many years.

Examination revealed that there was a slight ptosis of the right eye, which was in external strabismus. The pupils were equal and optic nerves normal. The tongue deviated toward the right side, and the right face was weak. The walk was pretty good, but hemiparetic. There was no talipes. There was marked atrophy in the extensor region of the right forearm, and when the forearm was flexed the hand and fingers were in the attitude called "cou de cygne," which has been already described. An attempt to extend the wrist causes flexion of the fingers, and *vice versa*. There are the usual hemiplegic reactions to electricity, and there is no anæsthesia. The hand is much smaller than its fellow and is very weak.

The next spasmodic deformity considered will be hysterical contracture. Like all other hysterical manifestations it conforms to no regular laws. The more common form resembles, in a great many particulars, a hemiplegic contracture. The forearm is generally firmly flexed and supinated. The hand is sharply flexed, more toward the ulnar side of the forearm. The thumb is inverted and the fingers tightly clenched into the palm of the hand.

Forced extension of the fingers is accomplished with great difficulty and with apparent pain to the patient, and when they are released they instantaneously return to their former position. The contracture can also be reduced by very strong faradization of the extensors, by static electricity, and by the magnet, as shown in the case submitted later. There is no atrophy, no trophic change, no loss of electro-contractility in the hand or forearm. There is, however, often a local or hemi-anæsthesia, achromatopsy, and loss of the senses of smell and taste on the same side with the contracture.

The history often reveals other hysterical manifestations, as convulsions, ovarian symptoms, globus, etc., etc., which, together with the other symptoms, will serve to distinguish an hysterical contracture from any other.

Sometimes there is contracture of one finger or the thumb only, as in a case reported by Dr. Adam. The case occurring in Charcot's service at the Salpêtière, and so ably treated and reported by Dr. Vigouroux, will be given in detail, as it is typical.

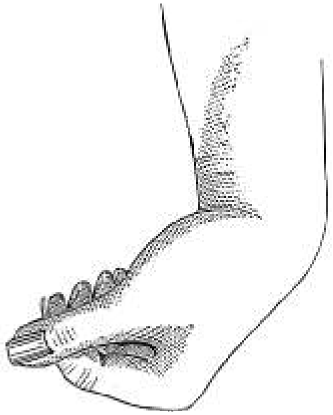

FIG. 18.—Left hand of Pauline J., Professor Charcot's case of hysterical contracture.

The patient was Pauline J.,[1] twenty-six years old, of large frame, ruddy complexion, muscular, and rather masculine in build, and left-handed. There were no antecedents. She began to menstruate at seventeen. The menses were always regular, but preceded by lumbar and hypogastric pains. July 17, 1874, while menstruating, sat in a cold place when overheated, and had a violent chill, accompanied by general hyperæsthesia. She was put to bed, when a violent headache came on. The chill lasted two hours, but the sensation of cold continued till a fever, with delirium, came on, which lasted eight days. The headache lasted fourteen days. After the headaches came hypogastric pains, with a sense of constriction, accompanied by retention of urine. The first attack lasted forty-eight hours, and these attacks occurred frequently in the next three years, four or five times. This attack was replaced by attacks of incessant vomiting. In November, 1877, one evening, she felt a numbness of the left hand, and on trying to raise it it fell inert. That night a sort of coma came on and lasted three days, preceded by intense headache. Nine days of somnolence, with headache, followed. The flaccid left hand soon began to grow rigid, and in three weeks it was closely shut. The patient had noticed a feebleness of the left leg, also; she had become emotional and had acquired a globus. She never, however, had any regular hysterical attacks.

On admission,[2] June 3, 1878, the left hand was flexed at a right angle, and the fingers were tightly flexed on a roll of linen held in the palm. The elbow and shoulder joints were mobile, but could not be voluntarily moved. There was no atrophy, and farado-contractility was retained. There was complete anæsthesia of the entire left upper extremity. The left half of the face was analgesic. The left ear, affected with tinnitus, was deaf. The left eye perceived colors, but had diminished acuity of vision. Smell was diminished and taste abolished on the left side. Pressure on the vertex and all down the spine was very painful. There was pain—made worse by pressure—in both ovarian regions.

Suffice it to say that various experiments were tried with the electro-magnet, solenoid, common magnet, galvanic, faradic, and static electricity, and finally a course of treatment was commenced by repeatedly producing a contracture on the right side with a strong magnet, which finally left the patient, July 23d, with no contracture, no anæsthesia, and with slight voluntary movements of the left fingers and wrist.

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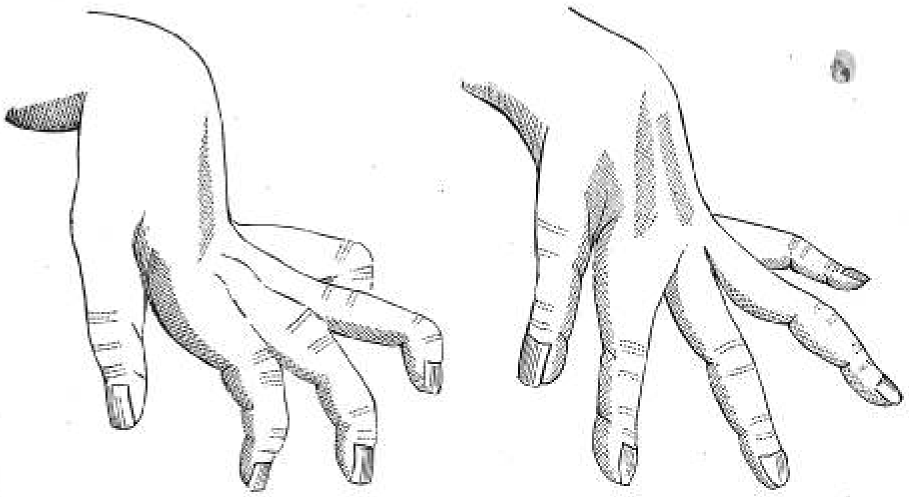
[1] Contracture hystérique du poignet gauche. Traitement par la production artificielle répétée d' une contracture du poignet droit. Disparition de la contracture primitive. Applications variées de l' electricité. Par le Dr. Romain Vigouroux. *Prog. Méd.*, 31 aout, 1878, p. 679, *et seq.*

[2] Service of Professor Charcot.

The ultimate recovery of the left upper extremity was apparently perfect. In the spring of 1880, however, the contracture and anæsthesia reappeared, and the patient returned to the clinic of Professor Charcot for treatment, and it was at that time (May 10, 1880) that the accompanying drawing was made. There was anæsthesia of the left upper extremity, no voluntary movements below the elbow, no atrophy, flexion of wrist to about a right angle, and tight flexion of the fingers and thumb on a linen compress in the hand. This contracture could not be overcome by any force safely applied to the fingers, but could be readily overcome by strong faradization to the extensors of the hand and fingers in the forearm. When last seen this case was under treatment by faradic and static electricity, and the occasional application of the magnet, but had not yielded, as before, to any treatment.



FIGS. 19 and 20.—Right hands of two patients of Professor Charcot's, with athetosis.

Athetosis is another condition which can be classed with contractures as a spasmodic deformity of the hand. Athetosis is not always a sequel to hemiplegia. It is confined to the extremities of one side, however, and appears generally after an apoplectic attack, a hemi- or general spasm, a severe headache, an aphasia, a vertigo, or some other manifestation of serious cerebral disturbance. Some weeks or months after such manifestations, which generally are hemiplegic, it is noticed by the patient that he is unable to keep his fingers or toes still, and that when left to themselves, and sometimes in spite of him, they are continually in motion. These movements are slow and vermicular, resemble in no particular any other spasm or any voluntary movement. They consist in alternate flexion and exaggerated extension, abduction and adduction of the fingers and thumb, all usually being meanwhile kept straight, the movement chiefly occurring at the metacarpo-phalangeal articulation. The more common attitude is extension and wide separation of the fingers and thumb. These movements are continuous, and only in a few cases can be entirely stopped by an effort of the will. The movements are aggravated by

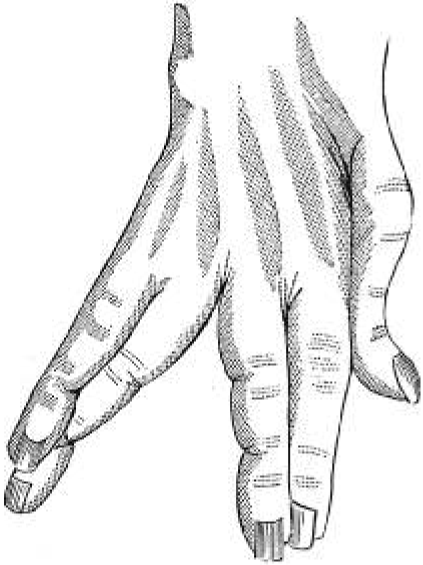


FIG. 21.—Right hand of a peculiar case of hemiplegia, followed by athetosis in the adult. Service of Professor Charcot.

an attempt to use the part or the opposite hand, and are accompanied by a good deal of pain. There may or may not be impairment of sensibility in the part, generally none.

Continual exercise often causes an hypertrophy of the affected muscles, so that the arm and leg of the affected side are often larger than the opposite, and of great hardness. There is, however, a real loss of voluntary power in the part. In many cases there occur occasional epileptic paroxysms, and there is generally considerable failure of the mental powers. There are seen no trophic changes in the parts, and no change in the electrical reactions of the muscles.

The above remarks sufficiently cover the clinical aspects of the disease, so it will suffice to say that the patients from whom two of the accompanying sketches were taken were idiots about thirteen years old, affected with infantile hemiplegia and partial epilepsy, in the service of Prof. Charcot, at the Salpêtrière (see figs. 19 and 20).

A peculiar deformity of the hand occurs in the course of the disease known as paralysis agitans. This disease, rarely seen, except in adults past forty, begins by a slight rythmical tremor generally in the hand, or the fingers of one hand. This tremor gradually increases, becomes more general in the extremity first attacked, and then invades the other extremity on the same side, and perhaps later the corresponding extremity on the opposite side, and even toward the last, though rarely, all four extremities. The exciting cause of the disease appears in many cases to be a mental shock of some kind. The movement is generally continuous during waking hours and absent during sleep; is made worse by excitement of any kind, and is aggravated by a depressed mental or physical condition. In almost all cases it can be temporarily stopped by a strong effort of the will. The continual motion causes no such hypertrophy as athetosis, and is very tiring to the patient, who often complains of severe pains in the muscles, which are the seat of tremor. The movement which is characteristic of the disease when fully developed is this: the arm is slightly abducted and the shoulder has a tendency to fall forward and inward on the chest, the forearm is flexed to nearly a right angle, thus bringing the hand in the neighborhood of the pubes, the most natural attitude for the patient. The hand is not generally flexed, but the fingers are semiflexed in a nearly straight condition, and the thumb is generally nearly opposed to them; the more common attitude then being that in which we hold a pen when writing. The fingers generally have the deviation toward the ulnar border of the hand, as in chronic rheumatism. Such being the position of the hand, now for the movements.

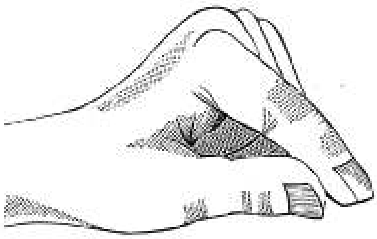


FIG. 22.—Left hand of a case of paralysis agitans under Professor Charcot's care.

Often there are slight flexions and extensions of the forearm and wrist. The fingers continually oscillate in movements of slight flexion and extension, which give the hand a pawing motion, and which, with opposition of the thumb, give the hand the appearance as if rolling a thread or paper ball between the fingers and thumb.

Later in the disease distortions like those in rheumatism may still further deform the hand, and atrophy may supervene from prolonged disease, and the electrical reactions may be modified, but in the uncomplicated disease they remain unchanged.

Late in the disease there comes on a marked rigidity of the spine, with flexion and projection forward of the head and neck, a fixed, stooping attitude, and a marked shortening of the stature. Slowness of speech and mental impairment often are present, and a symptom common to almost all is an unnatural subjective warmth of the body at night, or whenever they are in bed, leading them to require much less covering than other patients in the same ward, or than they themselves did before their sickness. The following is the history[1] of a typical case in which the tremor was very general:

The patient was a woman in the St. Alexandre ward of the Infirmary at the Salpêtrière under Prof. Charcot's care. She was 60 years old. Twelve years before she had a crying spell, brought on by some intense emotional disturbance. She noticed immediately afterward a weakness of the arms, first the right. Soon after the legs became weak, the right first. At the same time there were some cramps. Tremors appeared first in 1873.

The following notes were taken July 8, 1874:

The mouth is open about a centimetre ; the lower lip falls. There is some difficulty in deglutition. Her sleep is often broken by pains in the fingers ; she is always hot. The legs are adducted

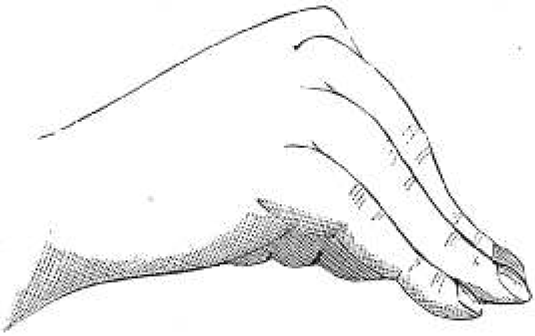


FIG. 23.—Right hand of a second case of paralysis agitans under Professor Charcot's care.

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[1] Taken partly from the hospital records.

and the trunk strongly bent forward. The arms are slightly abducted and flexed, so as to bring the hands into the subumbilical region. The thumb is slightly flexed, and the index is semiflexed, and the other fingers are semiflexed together. The head is fixed and rigid.

At the time the sketch (fig. 22) was made, June 19, 1880, the condition of the patient was little changed. She had not walked for several years, and could not raise herself in bed. She is taken out of bed and sits doubled up in a chair. The head is inclined to the left shoulder and slightly forward, and is fixed. There is some tremor of the lower lip and tongue when protruded. Her arms are slightly abducted, the elbows are flexed, and both hands rest in the lap near the pubes, and are in continual motion, of the ordinary pawing variety. There is ulnar deviation of the fingers and a contracture of the hand, which can be overcome by force. There is considerable atrophy of the first interosseous muscle. There is considerable tremor of the left foot. The parts which are the seat of spasm are also painful, and not only is sensibility retained, but there appears to be some hyperæsthesia of the legs.

3.—PARALYTIC DEFORMITIES.

Under the head of paralytic deformities, of course, is included the temporary condition of the hand immediately after various nerve injuries. This is especially true of the deformity produced by injury to the musculo-spiral nerve. This deformity is simply a drop wrist, and this is to be spoken of immediately under lead paralysis, where all the points in differential diagnosis will be fully brought out.

A person after exposure to lead in various ways, by inhalation, by swallowing, and in all probability sometimes endermically, and commonly after some other toxic manifestations of the poison, as colics, constipation, cachexia, etc., rather suddenly notices an inability to extend the wrist and fingers, generally on both sides. Examination reveals the existence of drop wrist. There is partial or complete paralysis of the extensors of the hand and fingers.

Unlike the drop wrist from injury to the musculo-spiral nerve the supinator longus escapes in saturnine paralysis. The electrical reactions are, however, much the same, very soon showing the degeneration reaction to galvanism and a loss of farado-contractility.

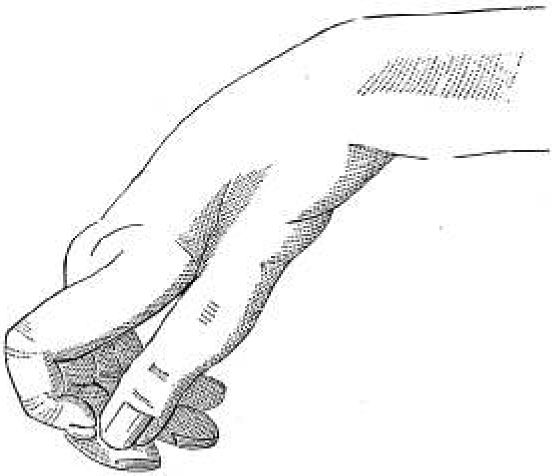

FIG. 24.—Right hand of case of plumbism, characterized by "drop wrist" and atrophy, under Dr. Seguin's care.

There is very apt to be a tumefaction on the back of the hand in these cases at first. If the paralysis remain long without treatment, atrophy may take place in the paralyzed area. There are no trophic changes, and there is generally only slight impairment of sensibility. As in the case here presented the paralysis is not always limited to the extensors.

Male, aged 42, first seen at the clinic of Dr. Seguin in the fall of 1878.[1] In July of that year, while he was occupied as cook on a freshly painted yacht, he woke up one morning with loss of power in both arms. He said he was in the habit of drinking water just from the tap every morning, and that this water was conducted through a lead pipe from the tank.

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[1] Part of the history was taken from the records at the College of Physicians and Surgeons.

On Sept. 20th, when first examined, he had double drop wrist, some flattening of the thenar and hypothenar eminences, and preservation of the supinator longus. He had had no colic, and there was now no blue line on the gums.

Oct. 12th there was noticed for the first time a swelling on the back of the wrists.

Three years later he again came under observation. The drop wrist still persisted. There was much atrophy of the thenar eminences and of the first interosseous space. The grasp was good. Extension of the two last phalanges was possible in all fingers except the right index. There was some adduction but no opposition of the thumb possible. They were inverted and their distal phalanges were flexed. There was some impairment of sensibility in this case, and degeneration reaction in all the paralyzed and atrophic area.

A deformity of the hand which may supervene after an infantile hemiplegia, besides the permanent contracture already spoken of, is that which is called retarded development. This hand, which has little of the element of spasm or contracture about it, is essentially a paralytic deformity. The hand is simply smaller and less developed than its fellow. It is well nourished, mobile, properly shaped, has no atrophy, and generally no anæsthesia. It is very weak, and perhaps the occasional seat of associated or athetoid movements. Its electrical reactions are normal. Sometimes, as in the case furnishing the subject for the accompanying sketch, there is a slight tendency toward contracture, but it offers no resistance and the part is generally very limp.

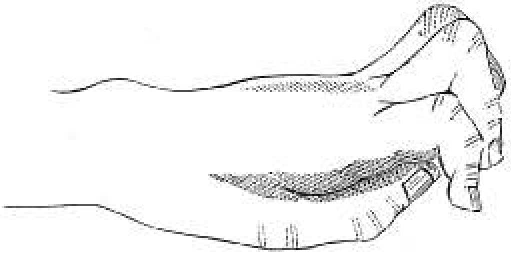

FIG. 25.—Left hand of K. B., patient of Dr. Seguin. Old hemiplegia and retarded development.

K. B.,[1] a girl of fourteen years, had a hemiplegic attack at the age of three. She never fully regained power on the left side, though at the time the sketch was made, June 8, 1881, the walk was hardly hemiplegic. The most noticeable feature in her case is the weakness and small size of her left upper extremity. The hand is perfectly shaped, and there is no atrophy, but it is very much smaller, as the following measurements will show :

	HEALTHY SIDE.	PARALYZED SIDE.
Circumference of the the wrist . . .	16. cm.	12.5 cm.
Circumference of the hand below the first phalanx of the thumb . . .	27.5 "	17. "
Circumference of the hand at the head of the metacarpal bones, excluding the thumb	19. "	15. "
Length of medius	10. "	8.5 "
Circumference of its first phalanx . .	5.75 "	5. "
Circumference of the thumb . . .	6.25 "	5.25 "

At times, as for instance when this sketch was made, there is a slight tendency to contracture in the hand of this patient.

4. DEFORMITIES OF LOCAL CAUSATION.

An exhaustive description of hand deformities of local causation would hardly be in place in a journal devoted to neurology, however important they might be. Brief mention, therefore, of a few will be made, illustrated as well as may be by sketches. Arthritis deformans (the chronic progressive articular rheumatism of Charcot), as a disease by itself, is seen chiefly in people of adult life. It quite often begins in peripheral parts and tends in a centripetal direction. It is very slow in its progress, and its course is marked by very many intermissions. It often commences by a painful swelling of one or more finger or toe joints. The swelling is not accompanied by much heat, redness, and no subsequent desquamation, as in gout. The joints implicated are apt to be symmetrical, and the inflammatory process leaves them more or less anchylosed and distorted.

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[1] Service of Dr. Seguin at the Manhattan Hospital.

Successive attacks recur in the same joints and implicate new ones, until a more or less complete anchylosis and marked deformity result. The disease, when advanced, implicates the larger joints of the body, even those of the spinal column, everywhere causing anchylosis and deformity. There are not always marked bony deposits about the diseased joints and nerve deposits outside the joints, as in gout. In most all cases, as a result of combined muscular action and bony deposit in the joints, there is a deviation of the fingers toward the ulnar side of the hand. Beyond that there are no very typical deformities. There is often seen an extension of the first phalanges, a flexion of the second, and an extension of the third. (See fig. 26.) In others all the phalanges are more or less flexed. (See fig. 27.) The thumbs generally escape. The course of the disease is frequently marked by œdematoüs swellings of the hands.

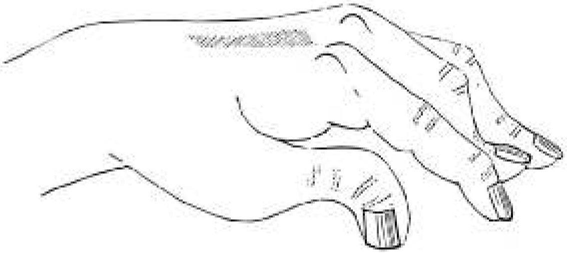

FIG. 26.—Left hand of a patient with arthritis deformans under Professor Charcot's care.

From disuse and possibly from some spreading of the disease to the nerves or muscles very marked atrophy of

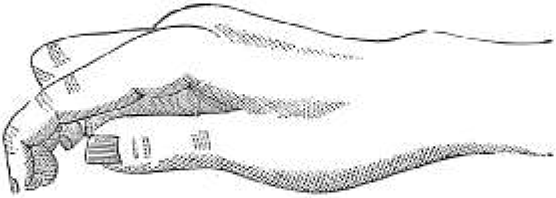

FIG. 27.—Right hand of a case of arthritis deformans under Dr. Seguin's care.

the intrinsic muscles of the hand ultimately ensues. Subluxation of the phalangeal joints sometimes occurs, and remarkable thinning and smoothness of the skin often ensue.

In rheumatic arthritis and sometimes accompanying acute articular rheumatism deformities of the hand are found, but they are not very typical. The disease is not always symmetrical; a single joint may be affected for years and a cure sometimes results. The inflamed joint is swollen, hot, and red, and very tender and painful. In almost all these particulars the disease differs from arthritis deformans.

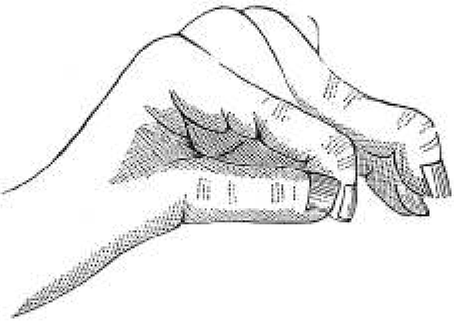

FIG. 28.—Left hand in cases of rheumatic arthritis under Prof. Charcot's care.

The deformity it leaves resembles that of arthritis deformans (see fig. 28) by presenting generally an ulnar deviation of the fingers and more or less osseous deposit about the joints. It differs again by often invading the thumb, which it leaves with extension of the last phalanx, giving it a curved appearance represented in fig. 29.

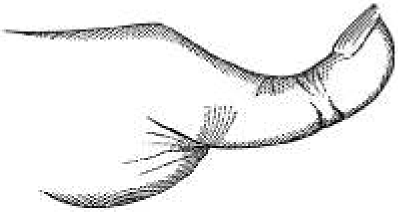

FIG. 29.—Left thumb in cases of rheumatic arthritis under Prof. Charcot's care.

The gouty might easily be mistaken for the rheumatic hand, particularly that form consisting simply of articular enlargements, anchylosis, and ulnar deviation of the fingers, were it not for the typical history the gouty case presents. When, however, the hand presents also the characteristic collections of urate of soda, or tophi, the deformity can be confounded with no other. These tophi present globular swellings, situated on various joints, varying in size from a

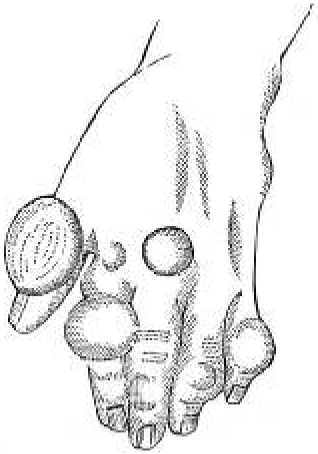

FIG. 30.—Gouty hand with tophi. Taken from Meillet.

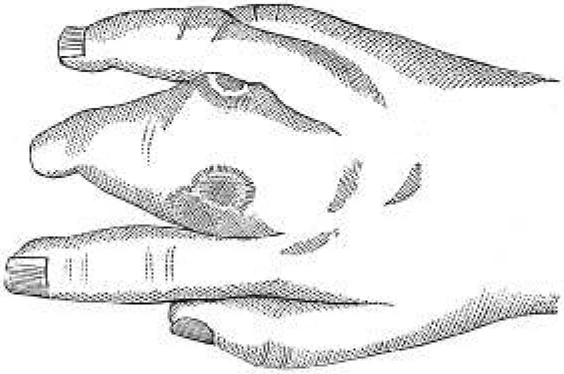

FIG. 31.—Hand with syphilitic dactylitis. Taken from Bumstead and Taylor's "Pathology and Treatment of Venereal Diseases." Phila., 1879.

pea to a pigeon's egg, covered by a thin bluish skin, often surrounded by tortuous, hardened veins. The indolent ulcers into which these concretions often break down would also be a mark of diagnostic value. (See fig. 30.)

The deformities produced by syphilitic dactylitis resemble in some features those of gout or rheumatism, but the clinical history, with the anatomical characteristics, will serve to distinguish them.

The deformity resulting from contraction of the palmar fascia cannot possibly be mistaken for any thing else, and all that is necessary is to introduce a cut copied from Meillet.

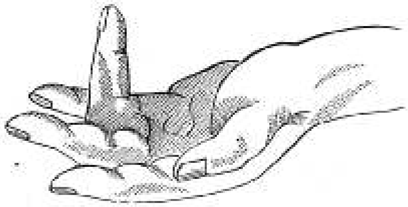

FIG. 35.—That of contraction of the palmar fascia, all taken from Meillet.

The bulbous finger tips and curved nails of cyanosis and phthisis need only figures borrowed also from Meillet.

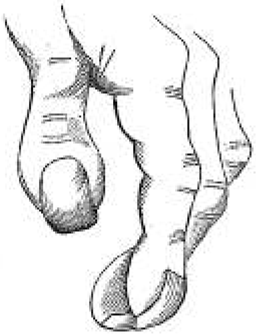

FIG. 33.—That of cyanosis.

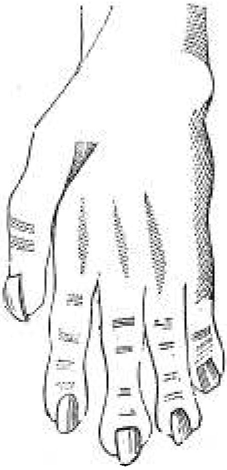

FIG. 34.—That of phthisis, and

The writer having seen but one case of scleroderma, and not having at command a sketch of her hands, borrows another illustration from Meillet, and regrets that space does not allow a brief *résumé* of that interesting disease.

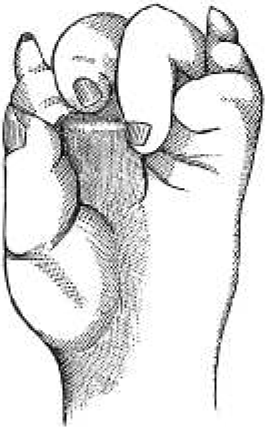

FIG. 32.—The hand of scleroderma.

At some future date a separate monograph may be made to treat more exhaustively of deformities of the hand of local causation, and perhaps those of a surgical nature, all of which are necessarily crowded out of an article like this, which treats simply of "medical" deformities.